WEST Search History

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DATE: Sunday, January 25, 2004

Hide?	Set Name	Query	Hit Count
		DB=USPT; PLUR=YES; OP=OR	
☐	L6	L5 and (smooth muscle same cell same proliferation)	30
☐	L5	double same balloon same catheter same gene	40
☐	L4	L2 and (smooth same muscle same cell same proliferation) and 4824436	35
☐	L3	L2 and (smooth same muscle same cell same proliferation)	107
☐	L2	l1 and angioplasty	426
☐	L1	double same balloon same catheter	708

END OF SEARCH HISTORY

h e b b cg b chh e e f ff e ch e

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=> s p21 gene
L1          1637 P21 GENE

=> s l1 and gene therapy
L2           123 L1 AND GENE THERAPY

=> s l2 and py <= 1995
   2 FILES SEARCHED...
   4 FILES SEARCHED...
L3             4 L2 AND PY <= 1995

=> dup rem
ENTER L# LIST OR (END):l3
PROCESSING COMPLETED FOR L3
L4             4 DUP REM L3 (0 DUPLICATES REMOVED)

=> d l4 tot ibib abs
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1720, 27

L4 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1996:51911 CAPLUS

DOCUMENT NUMBER: 124:114236

TITLE: Adenovirus-mediated over-expression of the cyclin/cyclin-dependent kinase inhibitor, p21 inhibits vascular smooth muscle cell proliferation and neointima formation in the rat carotid artery model of balloon angioplasty

AUTHOR(S): Chang, Mark W.; Barr, Eliav; Lu, Min Min; Barton, Kevin; Leiden, Jeffrey M.

CORPORATE SOURCE: Departments Medicine, University Chicago, Chicago, IL, 60637, USA

SOURCE: Journal of Clinical Investigation (1995), 96(5), 2260-8
CODEN: JCINAO; ISSN: 0021-9738

RBI. J5

PUBLISHER: Rockefeller University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vascular smooth muscle cell (VSMC) proliferation after arterial injury is important in the pathogenesis of a no. of vascular proliferative disorders, including atherosclerosis and restenosis after balloon angioplasty. Thus, a better understanding of the mol. mechanisms underlying VSMC proliferation in response to arterial injury would have important therapeutic implications for patients with atherosclerotic vascular disease. The p21 protein is a neg. regulator of mammalian cell cycle progression that functions both by inhibiting cyclin dependent kinases (CDKs) required for the initiation of S phase, and by binding to and inhibiting the DNA polymerase .delta. co-factor, proliferating cell nuclear antigen (PCNA). In this report, the authors show that adenovirus-mediated over-expression of human p21 inhibits growth factor-stimulated VSMC proliferation in vitro by efficiently arresting VSMCs in the G1 phase of the cell cycle. This p21-assocd. cell cycle arrest is assocd. both with significant inhibition of the phosphorylation of the retinoblastoma gene product (Rb) and with the formation of complexes between p21 and PCNA in VSMCs. In addn., the authors demonstrate that localized arterial infection with a p21-encoding adenovirus at the time of balloon angioplasty significantly reduced neointimal hyperplasia in the rat carotid artery model of restenosis. Taken together, these studies demonstrate the important role of p21 in regulating Rb phosphorylation and cell cycle progression in VSMC, and suggest a novel cytostatic **gene therapy** approach for restenosis and related vascular proliferative disorders.

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Nature 366, 701-704, 1993

Genes & Dev, 1994, 1750-1758

J. Clinical Investigation 90: 2044-2049

PNAS 91: 10732-10736

L4 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1995:496403 BIOSIS
DOCUMENT NUMBER: PREV199598519953
TITLE: The p21 cyclin-dependent kinase inhibitor suppresses tumorigenicity in vivo.
AUTHOR(S): Yang, Zhi-Yong; Perkins, Neil D.; Ohno, Takeshi; Nabel, Elizabeth G.; Nabel, Gary J. [Reprint author]
CORPORATE SOURCE: Univ. Mich. Med. Cent., Dep. Intern. Med., Room 4520, MSRBI, 1150 West Medical Center Dr., Ann Arbor, MI 48109-0650, USA
SOURCE: Nature Medicine, (1995) Vol. 1, No. 10, pp. 1052-1056. ISSN: 1078-8956.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Nov 1995
Last Updated on STN: 29 Nov 1995

AB The **p21 gene** encodes a cyclin-dependent kinase inhibitor that affects cell-cycle progression, but the potential of this gene product to serve as a tumour suppressor in vivo has not been established. In this report, we show that the growth of malignant cells in vitro and in vivo is inhibited by expression of p21. Expression of p21 resulted in an accumulation of cells in G0/G1, altered morphology, and cell differentiation, but apoptosis was not induced. Introduction of p21 with adenoviral vectors into malignant cells completely suppressed their growth in vivo and also reduced the growth of established pre-existing tumours. Gene transfer of p21 may provide a molecular genetic approach to arresting cancer cell growth by committing malignant cells irreversibly to a pathway of terminal differentiation.

L4 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1995:942874 CAPLUS

DOCUMENT NUMBER: 123:329515

TITLE: In vivo **gene therapy** with p53 or p21 adenovirus for prostate cancer

AUTHOR(S): Eastham, James A.; Hall, Simon J.; Sehgal, Inder; Wang, Jianxiang; Timme, Terry L.; Yang, Guang; Connell-Crowley, Lisa; Elledge, Stephen J.; Zhang, Wei-Wei; et al.

CORPORATE SOURCE: Scott Dep. Urol., Baylor Coll. Med., Houston, TX, 77030, USA

SOURCE: Cancer Research (**1995**), 55(22), 5151-5
CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We introduced the gene for wild-type human p53 or p21, a crit. downstream mediator of p53-induced growth suppression, into a p53-deficient mouse prostate cancer cell line using a recombinant adenoviral vector (Ad5CMV-p53 or Ad5CMV-p21). Elevated levels of endogenous mouse p21 mRNA provided evidence for the functional activity of virally transduced p53. Functional activity of viral-transduced p21 was demonstrated through immunopptn. of cellular protein exts., which showed that the viral-transduced p21 assocs. with cyclin-dependent kinase 2 and was sufficient to down-regulate the activity of the cyclin-dependent kinase by approx. 65%. In vitro growth assays revealed significantly higher growth suppression after Ad5CMV-p21 infection compared to Ad5CMV-p53. In vivo studies in syngeneic male mice with established s.c. prostate tumors demonstrated that the rate of growth and final tumor vol. were reduced to a much greater extent in mice that received intratumor injection of Ad5CMV-p21 compared to Ad5CMV-p53. In addn., the survival of host animals bearing tumors that were infected with Ad5CMV-p21, but not Ad5CMV-p53, was significantly extended. These data suggest that Ad5CMV-p21 may be effective as a therapeutic agent for prostate cancer.